CLINICAL PROCEEDINGS

of the

CHILDREN'S HOSPITAL

WASHINGTON, D. C.

July 1951

VOLUME VII NUMBER 8

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CLINICAL PROCEEDINGS

OF THE CHILDRENS HOSPITAL

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CONTENTS

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FREDERIC GERARD BURKE, M.D.

DEDICATION

This issue of CLINICAL PROCEEDINGS OF THE CHILDREN'S HOSPITAL is dedicated to Frederic G. Burke, in honor of his recent accession to the position of Professor of Pediatrics, Georgetown University Medical School.

Dr. Burke was born in England in 1917 and matriculated at Seton Hall, graduating in 1938. He then went on to Georgetown University Medical School and graduated in the class of 1942. This was followed by an internship at Georgetown University Hospital for one year and by a residency at Children's Hospital for two years. During this time he was a teaching fellow at George Washington Medical School from 1943 to 1945 and subsequently was made Director of Pediatric Instruction at both George Washington and Georgetown University Medical Schools from 1945 to 1949. In 1949 he was appointed Assistant Clinical Professor of Pediatrics. This was followed in 1950 by his promotion to Professor of Pediatrics and Head of the Department at Georgetown Medical School. Dr. Burke thus becomes one of the youngest heads of a pediatric department in the country. It might be added that his current position is richly deserved.

Dr. Burke's interests have been many and varied. He has been associate roentgenologist at Children's Hospital from 1944 to the present time, and is perhaps the only pediatrician in the country who likewise has performed full duties in a pediatric x-ray department. Similarly he has shown a keen interest in clinical investigation and during the past five years has been a co-author of some twenty medical publications. In spite of Dr. Burke's manifest activities, he has found time to raise a vigorous family, and is currently the father of four young children, ranging in age from six months to four years.

We all join in wishing Fred Burke the very best success in his new post, and feel thoroughly assured that he will turn in his usual splendid performance.

LEAD POISONING

Salient Features in Its Diagnosis and Treatment*†

Hobart T. Feldman, M.D.

In perusing the hospital records to compile some information about any one of many serious diseases for which children are admitted to the Children's Hospital, the infrequency of diagnosed cases of lead intoxication was readily apparent. The rarity of this diagnosis at our hospital stimulated an investigation of the literature to determine the incidence of lead poisoning in other institutions throughout the country. The information thus obtained offers suggestive evidence that we are inclined to forget that such an entity exists. This is borne out by comparing the total number of cases of lead intoxication so diagnosed in this hospital with those reported from other sources. Table I lists the series of cases of lead poisoning reported by various hospitals throughout the country within the past 18 years. The inclusive dates during which the cases of each series were collected are indicated whenever possible; the date otherwise listed is the year in which each series has been reported. The rarity of this entity at this hospital becomes even more apparent when one considers the fact that our dispensary out-ranks all others in the country in the number of annual out-patient visits. The fact that Washington, D. C., is less industrial than other cities should not be considered a valid reason for the few cases we have diagnosed; the concern here is not with the lead industry in adults, but rather with the incidence of lead poisoning in children, who are all exposed to the same hazard of ingesting or inhaling lead in and around the home.

Sources of Lead

The ingestion of paint continues to be the most common source of lead intoxication in children. There is no legal restriction of the amount of lead in paint manufactured for outdoor or indoor use. Almost all exterior house paint contains white lead (lead carbonate) in amounts up to 70 per cent. Interior wall paint, furniture paint and enamels are usually free of lead, but the deep-toned green and yellow paints used in interior decorating sometimes contain considerable amounts of lead chromate. In 19 states there are labelling laws[7] requiring that the amount of lead in the paint be specified on the can. *There is no such law in D. C.* For many years the manufacturers of cribs and children's furniture have recognized this danger, and

* Paper presented to the monthly Tuesday Luncheon Conference, April 10, 1951, Children's Hospital, Washington, D. C.

† Sponsored by E. C. Rice, M.D., Dept. of Pathology; S. Bessman, M.D., Dept. of Biochemistry; I. Lattman, M.D., Dept. of Roentgenology.

have used other, harmless pigments; however, some cribs are being repainted in the home with lead-containing paints. It is advisable to avoid yellow and green paint unless the label states that it does not contain lead.

Other sources of lead are less commonly found in the home today than they were a few years ago. In 1940, after many cases of poisoning in infants were reported from the use of lead nipple shields, a law was passed forbidding the manufacture of this product. A few proven cases have been traced to the ingestion of wax crayons[8] and the inhalation of chalk dust which contained lead in the coloring pigment.[9] White blackboard chalk itself is made of gypsum (plaster of Paris), and contains no lead. Lead poisoning has been reported in Orientals inhaling or ingesting face powder, but the only lead used in the manufacture of cosmetics in this country is occasionally found in certain hair dyes.[7] Steinberg reported one case of lead poisoning in a child who had inhaled the fumes from his brother's toy smelting

TABLE I

HOSPITAL	CASES
Children's Hospital, Boston (McKhann[1]), 1924–1933	89
Johns Hopkins (Ennis[2]), 1950	47
Harriet Lane Home (Ford-Holt[3]), 1944	36
Children's Hospital, Philadelphia (Rapoport[4]), 1941	30
Johns Hopkins (Blackman[5]), 1937	22
Children's Hospital, Washington, 1936–1951	7

set.[6] The most frequent source of poisoning by inhalation in children has been in the burning of old storage battery casings for fuel in the home. Some porcelain and pottery contain minute amounts of lead in the surface glaze, and the only remote possibility of excessive ingestion of lead would be from their use as a food container for lemon juice, vinegar, or other acid for several hours.[7] Today lead toys are infrequently found in the home, and toys are painted with coal-tar colors certified safe by the food and drug administration. In some of the condemned houses in various cities the water may contain excessive amounts of lead because of the lead which was used in the piping. One case of poisoning from such a source was reported by Clark, in England, in a 4-month old infant who drank one cupful of water a day.[10] The maximum safe limit of lead in the drinking water is said to be 0.05 part per million. The maximum daily ingestion of about 0.5 milligrams of lead in food or drink is considered safe.[11]

Incidence

The incidence of lead poisoning in children varies with the age. The maximum number of cases occur between two to three years of age, but

some have been reported in early infancy and later childhood. Table II shows the age incidence in several series from different hospitals.

Rapoport[4] has pointed out the seasonal incidence of lead poisoning in children, and its relationship to the intake of Vitamin D and sunlight. Of 30 cases of proved lead encephalopathy, the onset of encephalopathic symptoms in 24 patients occurred between the months of May and September, and in 2 additional patients, in November and December. None of these 26 patients had received cod liver oil or other Vitamin D preparations. On the other hand, the remaining 4 cases occurred in the fall and winter, and had received an adequate Vitamin D dietary supplement. Blackman[5] likewise demonstrated the onset of symptoms of lead encephalitis, in 19 out of 22 cases, between the months of May and September. By corroborating these findings with the results of controlled animal experiments, *the*

TABLE II

Age Incidence

HOSPITAL	CASES
Children's Memorial Hospital, 18 mos.–4½ years, Chicago[6]	22
Johns Hopkins, 13 mos.–7 yrs.[5]	22
Boston Children's, 1 yr.–5 yrs.	89
Children's Hospital, 1 yr.–6 yrs., Washington	7

conclusion is that a Vitamin D-producing agent, either sunlight or cod liver oil, *increases the amount of lead absorbed from the GI tract* (but has no influence on the amount of lead entering the body through the lungs). The photosensitizing action of sunlight on porphyrins, which are present in increased amounts in patients with plumbism, may play a minor role in contributing to the severity of the disease. Of the 7 cases seen at our hospital, 1 was admitted in June, 3 in August, 2 in September, and 1 in January! Lead intoxication occurs with about equal incidence in males and females; the racial incidence is about the same.

Route of Lead in the Body

In children, the most common route of absorption of lead is by way of the gastro-intestinal tract. This is explained by the mannerism known as PICA, defined as a craving for unnatural articles, which are placed in the mouth. Less common portals of entry include the respiratory tract, the skin, and the conjunctivae. In children, the course of events usually begins with the swallowing of chips of paint, and, although each piece may in itself contain an innocuous amount of lead, the procedure may be repeated day after day, for months or years. A small amount of the lead salt, which

is soluble in the intestine, is absorbed by the upper duodenum; the remainder is not absorbed, and is excreted in the feces. The absorbed lead first enters the portal circulation, but the form, and the vehicle, in which it is transported have not been definitely established. Whether it is carried in the diphosphate form soluble in the plasma, or in the red blood cells,[12] it is nevertheless deposited, in a large degree, in the liver, and much of this lead is excreted with the bile, and never reaches the systemic circulation. The remainder of the lead passes into the systemic circulation, part of which is transported to the growing ends of the long bones, and the margins of the flat bones, and is deposited as the insoluble tertiary lead phosphate. These deposits are not generated rapidly enough during the early phases of lead poisoning to show up as the typical lead line seen on the X-ray.[13] The lead which does not go to the bones originally, is deposited in the kidney, pancreas, brain, peripheral nerves, and the liver, where specific cellular damage occurs. If dust containing lead is inhaled, the insoluble lead salts are dissolved in the respiratory secretions by the formation of lead protein combinations and are subsequently absorbed into the circulation.[8] Once the source of lead is removed, the lead which has been deposited in the bones and tissues is *gradually* excreted from the body by normal physiologic processes.

The rate of deposition of lead into the tissues and bones of the body, and its ultimate excretion is altered by variations in diet, infection, and acidosis. If the diet contains an adequate amount of milk, the lead will tend to be deposited more rapidly in the ends of the long bones. On the other hand, if the diet is deficient in milk, or, if there is an intercurrent infection with acidosis, the lead is more rapidly withdrawn from the bones and tissues into the circulation. It is the presence of these potential factors influencing the rate of absorbtion, deposition, and excretion of lead which have formed the basis for a rational therapeutic approach.

Symptoms and Signs

Acute lead poisoning in children is rare. When it does occur, it is caused by a sudden, overwhelming exposure to lead, such as in the inhalation of fumes from burning of storage battery casings or the accidental ingestion of lead salts. The onset of symptoms is abrupt, with nausea, vomiting and abdominal pain. These may be rapidly followed by muscular weakness or pain, or paresthesia. Hemolytic crisis may occur, and death is not uncommon within a few days.[14]

Chronic lead poisoning is more common in children, because they are more inclined to ingest small amounts of lead over a long period of time. Symptoms do not depend on the amount of lead absorbed at one time, but upon the amount absorbed over a given period of time and its subsequent

release from the soft tissues or bones. Therefore, the symptoms are commonly intermittent, with exacerbations being stimulated by any factor, such as infection or metabolic disorder, which mobilizes the lead that had previously been deposited in the bones and tissues. The history is frequently that of periods of well-being interrupted by periods suggestive of poisoning.

The onset of symptoms is frequently insidious, and may be mild in infants. Constipation alone may be present for months, and the mother may not feel that this is of sufficient importance to warrant a visit to the physician. The gradually increasing anorexia, loss of weight, pallor, irritability, and then, abdominal colic and vomiting are frequently the presenting symptoms. Aub has stated that the colic is due to increased tonicity of the smooth muscle of the intestine, and that lead works directly on this smooth muscle, possibly in addition to the effects of the sympathetic nervous system.[15] The typical spontaneous disappearance of colic and vomiting may lull the physician into a false sense of security of "good treatment." Such has repeatedly been the case in the reports from our files and from other institutions.

The most prominent and severe symptoms of chronic lead intoxication in children are those referable to the central nervous system. Of 36 cases of plumbism reported by Holt, 31 showed definite evidence of encephalopathy.[3] These symptoms may not appear until the disease has existed for a long time, or, they may appear suddenly in association with some secondary disturbance producing a rapid release of large amounts of lead. The onset of encephalopathic symptoms is usually relatively abrupt, with severe, and repeated convulsions. The convulsions are more often generalized, but may be focal, and sometimes, the focal attacks occur on both sides in irregular sequence. The temperature is usually normal, but may be slightly elevated. Transient palsies may follow each seizure. The convulsions are sometimes very difficult to control, and children often die in a series of violent attacks. Such was the case with a 1-year old patient who was admitted to this hospital in convulsions, and in spite of vigorous attempts to control the seizures, the convulsions could not be stopped, and he died 7 hours after admission. In the interval between convulsions, the child may be drowsy or even comatose, but states of excitement and delirium are also seen. In 22 cases of encephalopathy due to lead, Blackman reported that the onset of vomiting in 10 cases occurred within 3 weeks prior to the first convulsion.[5] In many cases, the convulsions were preceded by behavior changes, such as nervousness, irritability, listlessness, disobedience, and crying spells. Hemiplegia and other types of cerebral palsies are not infrequent between convulsive episodes. Encephalopathy may occasionally be manifested by the cerebellar syndrome, with cerebellar gait and ataxia,

by retrobulbar neuritis, optic atrophy[24] and cranial nerve palsies, with ptosis of the lids or strabismus.

Polyneuritis, alone, or complicating encephalopathy may occur, especially in older children. In contrast to the wrist drop and foot drop seen in adults, the paralysis is not so selective in children, and involves *all* the muscle groups, flexors and extensors.[4] The lower extremities are usually more severely affected than the upper extremities, and the distal muscles are involved more frequently than the proximal ones. Muscle cramps is a very common complaint, and this was possibly the reason for the primary diagnoses of osteomyelitis and tuberculosis of the hip in two of the cases of lead poisoning admitted to this hospital.

Lead Line

Severe lead poisoning often occurs with the absence of a lead line in the gums in children. It is never present in infants without teeth. It is frequently seen on the buccal, rather than the labial aspect of the gums, and often near the molar, rather than the incisor teeth.[16] It may exist as a group of small discrete brownish dots, rather than as a "line."

TABLE III

*Levels of Lead in the Blood and Urine**

	MGM./100 GMS.	MGM./LITER
Normal upper limit	0.06	0.08
Safe indefinite period	0.07	0.15

* From Belknap, E. L.[17]

Diagnosis

The diagnosis of lead poisoning in children is dependent not only on a history of pica and suggestive symptoms, but also on the results of laboratory and roentgen examinations. The most positive laboratory evidence consists of the demonstration of excess amounts of lead in the blood and urine. The technic for analysis of lead is difficult and time consuming, and the possibility of error is increased by the improper collection of specimens in unsuitable containers. The amount of lead in the urine parallels the amount of lead in transit within the body.[8] Table III shows the normal upper limits of lead in the blood and urine, as well as what is considered safe levels for short, indefinite periods. In the single case of lead poisoning, in our series of 7 cases, in which blood and urine lead determinations were made, the Army Medical Center Laboratory reported a blood level of 0.070 milligram per 100 milliliters, and a urine level of 0.34 milligram per liter,

both results greatly exceeding the maximum safe level. This patient had symptoms of vomiting and alternating constipation and diarrhea for six months prior to admission, and yet the diagnosis was not suspected until after the sibling had died from lead encephalitis!

Changes in Red Blood Corpuscles

The red blood cells exhibit several changes in lead intoxication, two of which occur with sufficient frequency as to be useful, though not definitive, diagnostic aids.[18] Both stippling and polychromatophilia are present, the sum of these two paralleling the reticulocyte count. In many laboratories, stippling is reported as the number of stippled cells per million red blood cells. This figure is obtained by counting the number of stippled cells in 50 oil immersion fields—there are normally 3 to 7 stippled cells in 50 fields—and multiplying this sum by 100.[17] Stippling is demonstrated in the

TABLE IV

Per Cent of Cases Showing Stippling

HOSPITAL	CASES	PERCENTAGE
Children's Memorial Hospital, Chicago[6]	22	81
Johns Hopkins[5]	22	100
Children's Hospital, Washington	7	42

Wright's stain used for ordinary differential smears. The hypochromic anemia, present in all cases of long-standing plumbism, is not specific. The percentage of cases showing stippling, in 3 different series, is shown in table IV.

It is not uncommon for the urine to contain increased amounts of protein, and occasionally, sugar will be present. The cause of glycosuria is not clear, although one might postulate that it is caused by direct damage to the kidney.

The spinal fluid may be normal, especially in those cases without encephalopathic symptoms, or, it may be under increased pressure, with greatly increased protein, positive Pandy test, and changes in the cell count from normal to 100 or more lymphocytes, the average increase being 30 to 40 cells. Ford[3] states that lead encephalopathy is always accompanied by definite changes in the spinal fluid.

The X-ray changes in the ends of the shafts of the long bones do not occur during the early phases of lead poisoning. When the child has ingested lead over a period of several months heavy transverse lines appear in the ends of the shafts, and sometimes in the ribs. During acute infections and metabolic disturbances, lead may disappear from the bones and reappear

in the circulation.[14] Thus lead poisoning, with all its clinical and laboratory manifestations, may exist in the absence of lead line in the bone. Nor does presence of this line of increased density, by itself, incriminate lead; similar findings are sometimes seen in normal patients, as well as in healing rickets and other metabolic diseases. Many patients exhibit X-ray signs of increased intracranial pressure, and occasionally, opaque, lead-containing material is visible in the flat plate of the abdomen, within the intestinal tract.[13]

Differential Diagnosis

The abdominal symptoms of lead poisoning have resulted in erroneous diagnoses varying from epidemic vomiting to mesenteric thrombosis. More than one case has had an appendectomy with temporary relief of symptoms.

TABLE V

Admission Diagnosis

	CASES
Tuberculosis of hip	1
Chronic Lead Poisoning	2*
Splenomegaly	1
Osteomyelitis	1
Convulsions	1
	7

* 1 case previously diagnosed at another hospital.

Acute intestinal obstruction, intussusception and Meckel's diverticulum may almost exactly mimic lead colic, with constipation, vomiting, blood in the stools, and rhythmical abdominal pain. Other diagnoses have included perforated peptic ulcer and pancreatitis.[17] An acute hemolytic crisis due to a blood dyscrasia, with hematuria, anemia and shock may be confused with lead poisoning.

The encephalitic symptoms, so common in children with plumbism, often result in the diagnosis of tuberculous meningitis. This is a natural mistake to make, according to Ford, especially if the tuberculin test is positive in a young child with stupor, rigidity, and increased protein and cells in his spinal fluid. Not infrequently, cases are misdiagnosed as behavior problems, brain tumor, or epilepsy. The polyneuritis of lead poisoning has been confused with poliomyelitis, Guillain-Barré Syndrome, and spinal cord lesions. The admission diagnoses of the 7 cases at this hospital are shown in table V.

Tuberculosis of the hip was suspected because the child complained of pain in the hip, and had been discharged from Glen Dale Sanatorium 6 months prior to admission to this hospital. The one case diagnosed as lead poisoning had been completely worked up at another hospital, and this admission was because of a behavior problem and vomiting. The case of splenomegaly was subsequently readmitted, and the correct diagnosis was made, after the history was obtained that the child's older sister had died of lead poisoning. This case was especially interesting because of the enlarged spleen, and has been reported as a new symptom-complex of lead poisoning by Dr. John McGovern.[19] The case of epilepsy, as well as the one of convulsions, complained of vomiting and convulsions. This last case died 7 hours after admission. The other 6 cases were discharged improved, but unfortunately, only 3 returned for follow-up examinations in the outpatient department.

Treatment

There is no universal agreement as to the treatment of chronic lead poisoning with specific therapy. Over 20 years ago it was pointed out that lead in the body is absorbed, transported, deposited, and excreted much as calcium. In other words, a positive calcium balance facilitates immobilization of lead in the bones, and a negative calcium balance results in its removal. A high calcium diet with milk and vitamin D enhances deposition. It is well known that intercurrent infections, such as pneumonia, diarrhea, etc., other causes of acidosis, iodides, parathyroid hormone, surgical operations, and alcoholism remove lead and calcium from the depot sites. This creates a continuous potential danger because accidental or incidental mobilization, by any of these conditions, is credited with the periodic acute exacerbations of chronic plumbism. The question is therefore whether one should plan a therapeutic course based on (1) deposition of lead in depot sites without subsequent deleading; (2) deposition of lead in depot sites, followed by subsequent carefully managed deleading or (3) deleading without previous planned deposition. The approach to this problem has been altered within the past few years by the discovery of several substances which are believed to mobilize the lead from the tissues and transport it in the form of a non-toxic, non-ionizable complex to the kidney, where it is excreted. One of these substances, known as BAL, or dimercaprol, is known to combine with other heavy metals, such as arsenic and gold, in the form of stable complexes in which they are excreted, and is thought to act much the same with lead. On the basis of this, many investigators have been using BAL, with supportive measures, in the treatment of lead encephalopathy.[2, 12, 21, 22] BAL is put up as a 10 per cent solution in benzyl benzoate and oil. Ennis used the dose schedule in table VI on 16 patients with lead

encephalitis; 7 were mild cases, 9 were severe. His conclusion was that BAL lowered the mortality and probably the severity of residua of severe lead encephalopathy. Some investigators are skeptical of the value of BAL in plumbism, because the lead-BAL complex is not so stable as other metal complexes, and the lead may return to a soluble form in the blood stream, resulting in increasingly severe symptoms. Sodium citrate is considered a safer and possibly more reliable therapeutic aid in deleading the patient, and it has been widely used, successfully, in both lead colic, and in the acute stages of lead encephalopathy.[15, 23, 24, 25] This salt dissolves the tertiary lead phosphate and combines with the lead to form a non-toxic complex[26] making lead available for excretion without raising the ionic level in the blood. If sodium citrate is used, the dose is 1 gram three times a day for a period of several months. Recently disodium hydrogen phosphate has been shown to be capable of safely deleading the body because

TABLE VI

BAL Dose Schedule (Ennis)

(10% Sol in Benzyl Benzoate and oil)
2 mg./kilo/inj. q4h x 4 doses, then
3 mg./kilo/inj. q4h x 4 doses, then
4 mg./kilo/inj. q4h x 4 doses, for 10 days

of its formation with the lead into a phosphate.[20] By this process, lead is mobilized and detoxified at the same time. The dose in adults varies from 5 grains three times a day for five days to 60 grains three times a day for fifteen days. In acute cases, it may be given in a 5 per cent solution intravenously (5–10 grains per dose) three times each day. In addition to these specifically acting drugs, every attempt should be made to control the encephalitic symptoms by the use of sedatives, and decompression by surgery, if medication does not control the convulsions.[2, 14] Hypertonic glucose and intramuscular magnesium sulfate, 50 per cent solution, 0.2 c.c. per kilo every 4 hours have been used many times, with not too much effect.

Prognosis

The prognosis of lead poisoning depends on the length of time the patient has had symptoms. If the mother of every child who is brought in to the office with the complaints of recurrent colic, vomiting, constipation, or encephalopathic-like symptoms is questioned specifically about the possibility of the child's ingesting lead from one of the several sources which may be present in the home, the diagnosis of lead poisoning should be seriously considered, and every effort should be made to corroborate these suggestive symptoms with the results of specific laboratory and roentgen

examinations. Even in patients who have manifested symptoms for a short period of time, and in mild, non-encephalitic forms, the prognosis should be guarded. Prior to sodium citrate, BAL, and disodium hydrogen phosphate therapy, the outlook was poor. In McKhann's series of 89 cases, 11 died of encephalitis, and permanent sequelae were present in many more. Mild lead poisoning in infancy may result in serious impairment of growth and nervous system development; often these do not become apparent until childhood.[2] Encephalopathy occurs in at least one-half of the infants and children with lead poisoning; the over-all mortality from this is well over 25 per cent.

In conclusion, it should be emphasized that although lead poisoning is much less frequent than many other diseases seen in the practice of pediatrics, it nevertheless exists today, and, because of the irreversible and often fatal sequelae which so frequently occur in unrecognized, untreated cases, it behooves the pediatrician to keep in mind the possibility of this poisoning. The diagnosis should rest on the presence of suggestive symptoms, a history of pica, the laboratory findings of excessive amounts of lead in the blood and urine, basophilic stippling, reticulocytosis, anemia, and the telltale lead line seen on the X-ray. The paucity of cases of lead poisoning recorded in the files of this hospital, as compared to the much greater numbers reported elsewhere, should stimulate us to look out for lead, or we will continue to miss it.

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ADDENDUM

Two additional cases of lead poisoning have been diagnosed at this hospital since the presentation of this paper.

CARDIOVASCULAR SURGERY IN CONGENITAL ACYANOTIC MALFORMATIONS

Special Report No. 210

Marshall C. Sanford, M.D.

Cardiovascular surgery is a new and rapidly-developing field. Operative procedures on some of the acquired conditions have been attempted in the past, but mortality was high; and results, often disappointing. The field of congenital cardiovascular surgery began in 1938 when Gross[1] first successfully obliterated a patent ductus arteriosus. As the result of the work by Blalock and Park[2] and Crafoord and Nylin[14] in 1944, and Gross and Hufnagel[3] in 1945, coarctation of the aorta was the next congenital cardiac anomaly to be successfully corrected. The most dramatic step forward in this field was the development in 1945 of shunting procedures in cyanotic conditions by Blalock and Taussig[4, 5, 6]—the first treatment of congenital *intracardiac* anomalies.

Improvements in anesthesia, chemotherapy, diagnostic aids, and new operative techniques are some of the factors responsible for these advances. The horizon continues to widen as more conditions fall within the scope of corrective cardiac surgery. The mortality and morbidity rates of these operative procedures lower steadily. All workers in this field eagerly await the day when a satisfactory heart-lung preparation will be perfected so that deliberate surgical procedures may be carried out on bloodless cardiac chambers.

Congenital cardiovascular defects are not rare in infancy; in contrast, the incidence after puberty is low because of the high early mortality. Until recently, most of these anomalies were of little more than academic interest. A veil of mystery still surrounds them. Therefore, it becomes apparent why many well-trained physicians feel dubious about managing such patients and are anxious to refer them to a specialist or to a large medical center.

It is the general impression among many physicians that extensive laboratory studies including cardiac catheterization and angiocardiography are routinely employed to diagnose the various cardiovascular conditions. Actually in the *typical* patient, the diagnosis is not difficult and one need only to employ the usual diagnostic procedures. In many of the acyanotic cardiovascular conditions, the diagnosis is established by physical examination alone. In dealing with the *less common or more bizarre anomalies*, extensive investigation is required and specialized techniques must be employed.

Certain basic studies are necessary in the majority of these patients. These include a careful history with emphasis on exercise tolerance and

the presence or absence of cyanosis, a thorough physical examination with particular reference to the nature of murmurs or thrills which may be present, fluoroscopy, roentgenographic examination of the chest (oblique views, when indicated), complete blood count, and electrocardiography. Barium study of the esophagus, lipiodol examination of the trachea, arterial oxygen saturation, angiocardiography, and cardiac catheterization may be necessary in special cases.

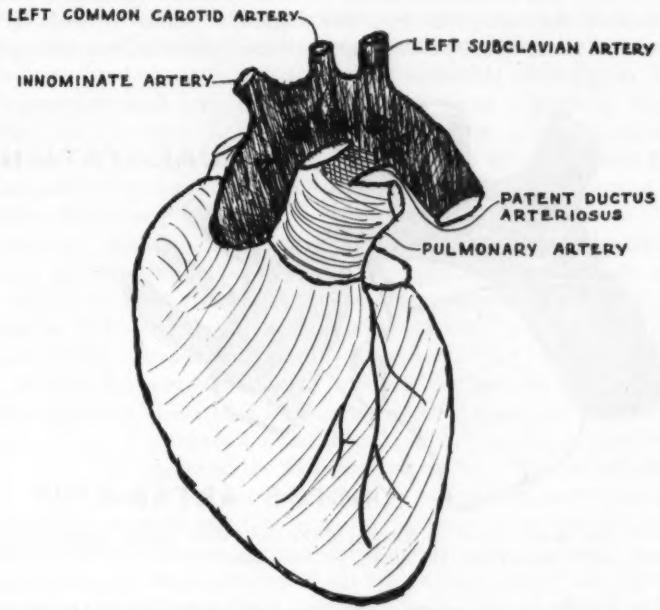

FIG. 1. Patent ductus arteriosus

This discussion will be limited to the more common congenital cardiovascular anomalies which are not associated with cyanosis and for which satisfactory surgical correction is well established. No attempt will be made to discuss the atypical or less common malformations or those for which the operative techniques are still in the experimental stages.

PATENT DUCTUS ARTERIOSUS

This is one of the most common acyanotic congenital cardiovascular anomalies. The ductus is a vessel which is given off from the pulmonary artery near its bifurcation and opens into the aorta beyond the origin of the left subclavian artery. The heart itself is not abnormal but the patent

ductus arteriosus persists beyond fetal life during which time it is normally functioning. At birth, with the expansion of the lungs, the pulmonary pressure falls and the need for the ductus ceases. Obliteration begins immediately and normally is complete within a few weeks or months.

In those cases in which the obliteration does not take place, an abnormal shunt is produced. Blood flows from the high-pressure aorta into the low-pressure pulmonary circuit. This shunt greatly increases the work of the left ventricle, and by increasing the pulmonary blood volume, it adds to the work of the right ventricle. The diagnosis usually is made by the existence of a characteristic blowing, machinery-like, continuous murmur

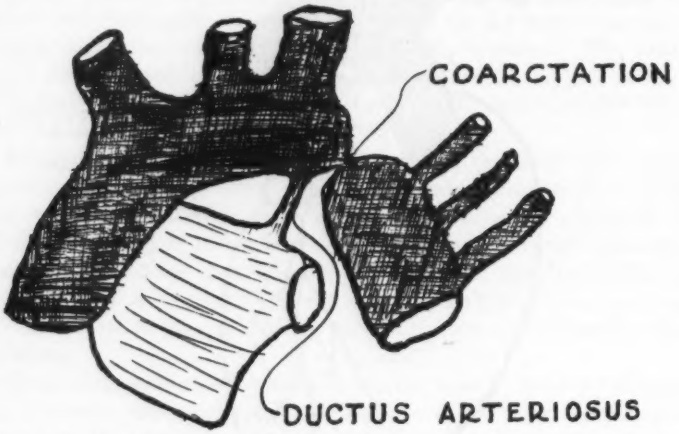

FIG. 2. The usual type of coarctation of the aorta with a short segment of vessel involved, and in most cases, the ductus is obliterated.

which is loudest in the second interspace just to the left of the sternum. A thrill may be present in this area. There is no cyanosis or clubbing of the fingers or toes. X-ray may reveal some fullness in the region of the pulmonary conus but this finding is not characteristic. The existence of symptoms depends on the size of the ductus and often these do not appear until late. Recently, with the aid of cardiac catheterization or angiocardiography, cases with patent ductus arteriosus without a murmur have been detected and confirmed at operation.

Obliteration of the ductus is indicated in all cases since the life expectancy in these patients is reduced. They are particularly susceptible to subacute bacterial endocarditis and of even greater importance, is cardiac failure resulting from the strain on the left ventricle. The optimal age for operation once was considered to be between four and ten years

of age. Now, after observing the damage produced by persistent high pulmonary pressure, earlier operation is being advised. Obliteration may be accomplished by division and suture[7, 8, 9] or by multiple suture technique without division.[10, 11] There are indications for both methods. Single or double ligation alone should be condemned since recanalization frequently results.

COARCTATION OF THE AORTA

This condition consists in a narrowing of the aorta of varying degrees in the region of the ductus arteriosus. Coarctation is usually discussed under two headings, although there is considerable overlapping. In the more common adult type, a short segment of vessel is involved, whereas, in the infantile type, the stenosis usually involves a longer segment of vessel and often is proximal to the origin of the left subclavian artery. In this latter type associated anomalies are common and most of these patients die in early infancy.

Diagnosis—Signs and symptoms of coarctation vary with the degree of stenosis and the size of the collateral pathways. Symptoms rarely appear before the second or third decade. The diagnosis is made on physical examination alone and should be seriously considered in all cases of hypertension. There is hypertension in the upper part of the body and hypotension in the lower part of the body. Normally the pressure in the legs is 20 to 40 millimeters higher than in the arms while with coarctation, the blood pressure is low or absent in the legs. Blood pressure should be taken in both arms. Often, it is higher on the right than on the left. Valuable information regarding the site of the coarctation can be gained from this examination. Evidence of numerous collateral pathways is found in the pulsating vessels which are most prominent in the pectoral and latissimus muscles. An x-ray of the chest may show notching of the ribs, which results from the pulsations of the dilated intercostal arteries against the lower margin of the ribs. This finding usually is not present before puberty. The femoral arterial pulses are diminished or absent. The heart most commonly is normal although a systolic murmur is not an uncommon finding. The position, intensity, and duration of murmurs are quite variable. It is important to obtain an electrocardiogram to determine the degree of myocardial damage. Although angiocardiography or aortography are not routinely employed, important information can be obtained from these studies.

Selection of Cases: All cases detected during childhood or early adulthood should be considered for operation unless definite contraindications to operation exist. After the patient has reached the fourth decade, the incidence of complications such as aneurysm, sclerotic changes in the wall of the vessel and the changes caused by prolonged hypertension cause one to

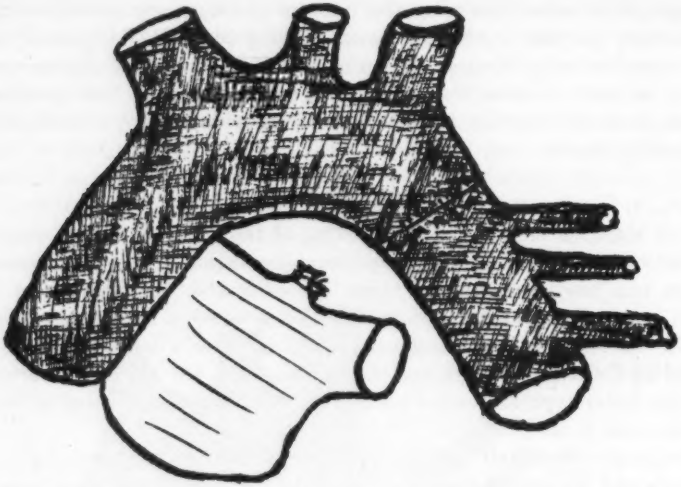

FIG. 3. The coarctation has been resected and an end-to-end anastamosis performed.

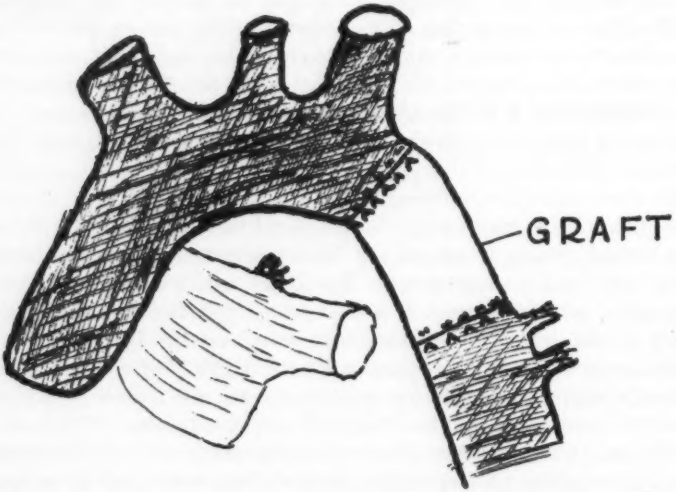

FIG. 4. One method of bridging a long gap utilizing a graft to connect the proximal and distal aorta.

investigate these cases more carefully and once the fifth decade has been reached, only the most exceptional case should ever be seriously considered for operation.

Prognosis without Operation: The need for surgical correction is appreciated if one analyzes the cases not treated by operation.[12] About one-fourth of them live a normal life with little or no incapacity. The others die from bacterial endocarditis, from hypertensive states, or from rupture of the aorta usually associated with aneurysm. In the hypertensive states, cardiac failure is twice as common as intracranial hemorrhage. The average age of death is about thirty years.

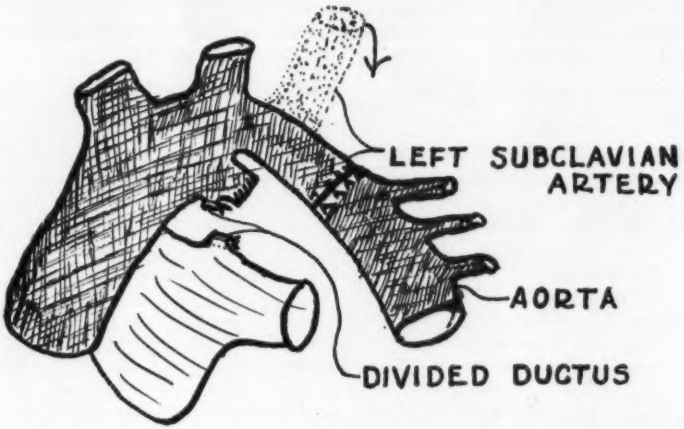

FIG. 5. The dilated left subclavian artery has been turned down and anastamosed to the distal aorta.

Operation: In March of 1944, Blalock and Park[2] first described an operation for the correction of this condition. They suggested bypassing the stenotic area by suturing the proximal end of the divided left subclavian artery into the aorta below the coarctation. In October of 1944 Crafoord and Nylin[14] of Sweden performed the first successful operation for the correction of this condition, and in July 1945 Gross and Hufnagel[3] in this country independently performed a similar procedure. They both resected the stenotic segment of the aorta between clamps and performed an end-to-end anastamosis.

Since that time large series of these cases have been reported.[6, 12, 13] The results are good; the mortality, low. The treatment of choice consists in a resection of the stenotic portion of the aorta with an end-to-end anastamosis. If the two ends cannot safely be joined together without excessive

tension, the gap may be bridged by an aortic graft[12, 15] or the dilated left subclavian artery may be turned down to join the distal aortic segment in an end-to-end union.

ANOMALOUS INTRATHORACIC VESSELS

Intrathoracic vascular anomalies are more common than is generally realized. They result from disturbances in the maturation process of the fetal vascular network. Most of them cause no symptoms and are accidentally discovered at operation or at autopsy. Venous anomalies may produce abnormal shunts and will be dealt with in a subsequent publication. Symptoms caused by arterial vascular malformations result from compression of the trachea or esophagus or both. It is extremely important to recognize these conditions during life so that proper therapy can be instituted.

Diagnostic methods are available and the most pertinent of these include:

1. *X-ray of the Chest:* (Anterior-posterior and lateral views). From these examinations, the amount and distribution of any existing pneumonia can be determined as well as the general position and contour of the trachea. Valuable information is gained from variations in the degree of aeration of the lungs during inspiration and expiration. If partial tracheal obstruction exists, areas of atelectasis may be seen on inspiration, while on expiration, emphysema may be noted.
2. *Barium Swallow of the Esophagus:* (Anterior-posterior and lateral views). Vascular anomalies may cause compression of the posterior or lateral walls of the esophagus. The lateral compression will be visible on the anterior-posterior view, while the posterior-wall indentation is best seen in the lateral x-ray. These deformities are more marked in the region of the third or fourth thoracic vertebrae.
3. *Tracheogram:* (Anterior-posterior and lateral views). Outlining the tracheal walls with lipiodol allows x-ray visualization of the compression caused by these various anomalies.
4. *Bronchoscopy and Esophagoscopy:* Direct examination of the lumen of both the trachea and the esophagus should be performed in all but the smallest infants. Information regarding the location and extent of any external compression can be obtained from these studies.
5. *Aortogram or Arteriogram:* (Using diodrast). Indications for this study are infrequent. The diagnosis is made by the above studies in most instances. If

FIG. 6. Typical x-rays from cases of double aortic arch. A. Lateral view, with large posterior compression of esophagus. B. Lateral view with smaller posterior indentation of esophagus. C. A-P view with moderate indentation of both sides of esophagus. D. Combined tracheogram and esophagogram. Trachea is quite narrowed while posterior wall of esophagus is compressed at slightly lower level. E. Visualization of trachea by lipiodol. There is compression of both sides of trachea. F. Lateral view of trachea, showing marked narrowing. (*From Robert E. Gross and E. D. B. Neuhausser: "Compression of theTrachea or Esophagus by Vascular Anomalies." Pediatrics, January 1951. Courtesy of Charles C Thomas, publisher, Springfield, Illinois.*)

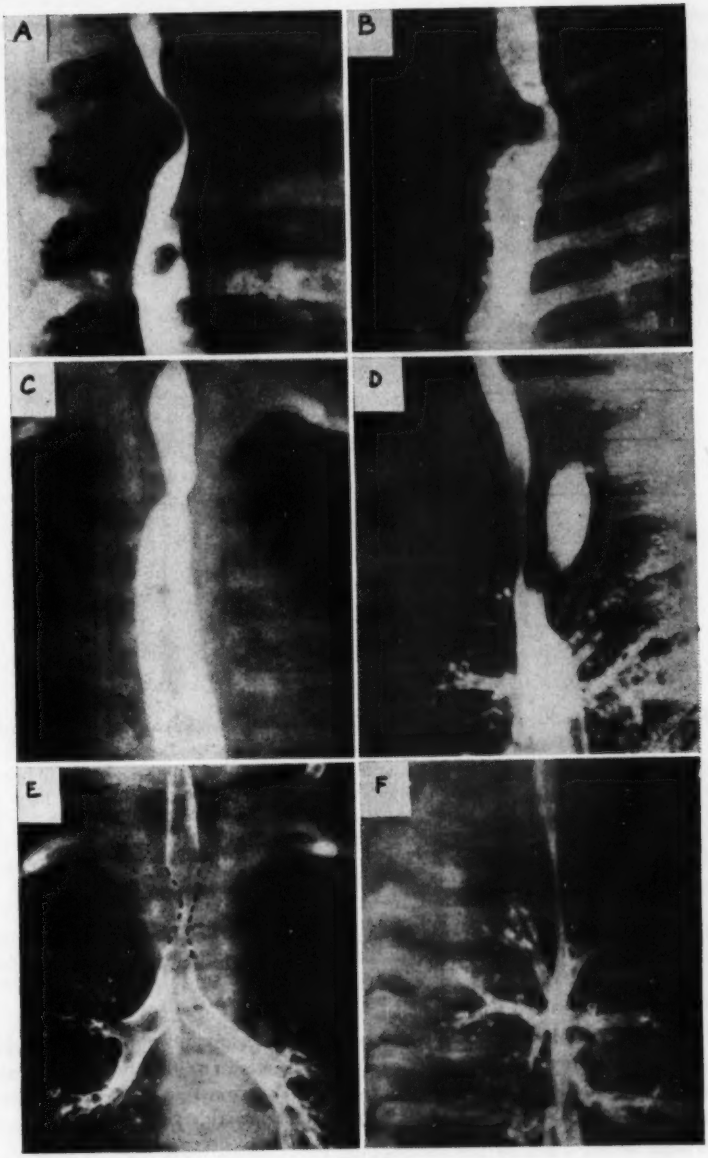

FIG. 6.

doubt remains, the aortic arch and its branches can be demonstrated by direct diodrast injection into the aorta, or by retrograde injection of one of its branches.

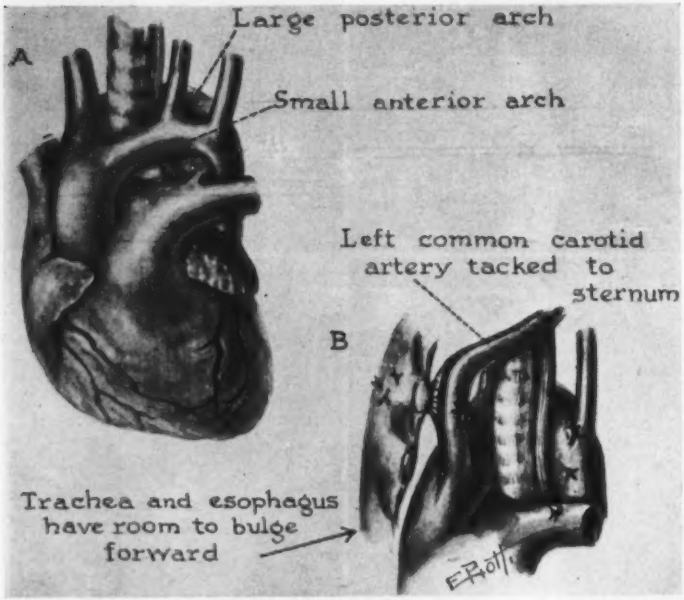

FIG. 7. Double aortic arch with large posterior limb and smaller anterior limb. Surgical alleviation of condition. Ligamentum arteriosum has been divided to allow pulmonary artery to fall forward. Small anterior aortic arch has been divided to break constricting ring. Left common carotid artery has been tacked forward to back of sternum, so that it will not press upon anterior surface of trachea. (*From Robert E. Gross and E. D. B. Neuhausser: "Compression of the Trachea or Esophagus by Vascular Anomalies." Pediatrics, January 1951, Courtesy of Charles C Thomas, Publisher, Springfield, Illinois.*)

The arterial vascular anomalies may be considered under the following headings:[6, 16, 17]

I. Double Aortic Arch

The usual course of the aorta is to ascend, pass anterior and to the left of the trachea and then proceed down the left side of the body. In a certain number of cases, the ascending aorta divides into two branches: one which passes in front of and to the left of the trachea while the other passes behind and to the right of the esophagus. Both limbs then join to form the

descending aorta. Rarely, the trachea only may be encircled, the posterior arch passing in front of the esophagus. In most instances, the anterior (left) arch is the smaller of the two. Not all patients with this condition

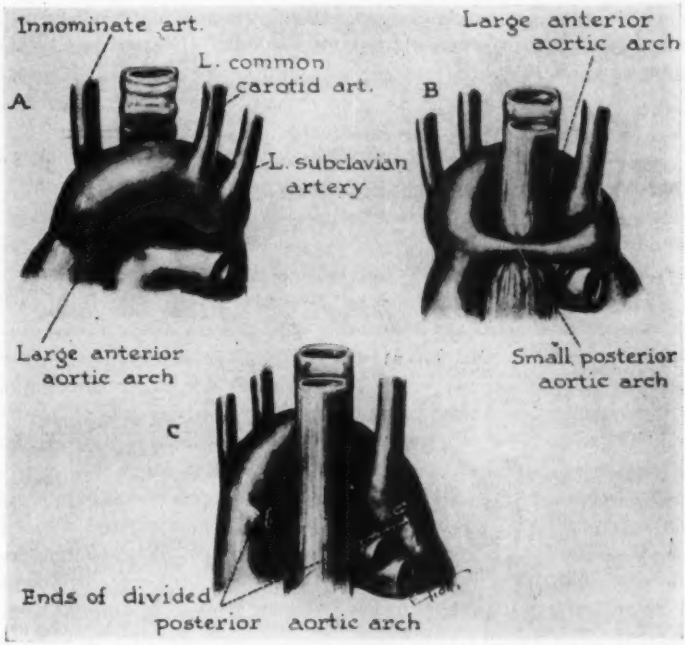

FIG. 8. Double aortic arch with large anterior limb and smaller posterior limb. A and B. Anterior and posterior views of anatomic arrangements, showing encirclement and constriction of trachea and esophagus. C. Surgical cure of condition by complete division of smaller posterior arch. (*From Robert E. Gross and E. D. B. Neuhausser: "Compression of the Trachea or Esophagus by Vascular Anomalies." Pediatrics, January 1951. Courtesy of Charles C Thomas, Publisher, Springfield, Illinois.*)

have symptoms, and the presence or absence of them depend on the amount of space between the two limbs and the encircled structures. This space usually is small and compression of the trachea or esophagus results. Gross[16] states that the average age of onset of symptoms is about six months.

This anomaly is not to be regarded lightly since it often causes death during the first two years of life. Death results from partial tracheal obstruction with superimposed pneumonia. The diagnosis should be sug-

gested by difficulty in swallowing, respiratory stridor, or recurrent pulmonary infections.

The diagnosis would be confirmed by x-ray evidence of tracheal distor-

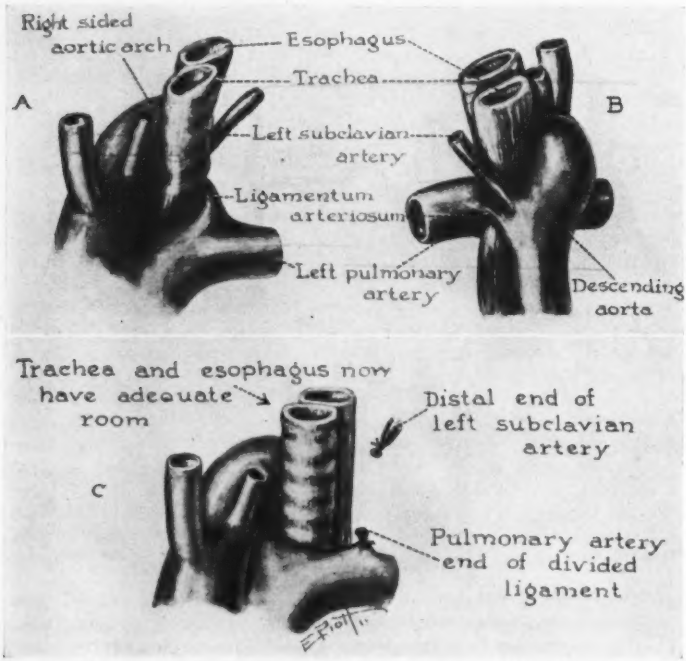

FIG. 9. Right aortic arch with left-posterior ligamentum arteriosus. A and B. Anatomic arrangements with constriction of esophagus and trachea. Left subclavian artery and ligamentum arteriosum sometimes arise close to one another from aortic wall and 2 taut structures have tendency to pull out aortic wall into sort of diverticulum. C. Surgical alleviation of anomaly. Ligamentum arteriosum and first part of left subclavian artery have been divided, thus allowing esophagus and trachea to displace posteriorly and to patient's left. (*From Robert E. Gross and E. D. B. Neuhausser: "Compression of the Trachea or Esophagus by Vascular Anomalies." Pediatrics, January 1951. Courtesy of Charles C Thomas, Publisher, Springfield, Illinois.*)

tion or compression, pneumonitis, and evidence of partial tracheal obstruction with the resultant inspiratory and expiratory changes. Barium swallow of the esophagus may show compression of both lateral walls or indentation of the posterior esophageal wall at the level of the third or fourth thoracic vertebrae. Arteriograms are rarely indicated. Tracheograms should dem-

onstrate the anterior constriction on the lateral view and bilateral constriction on the anterior-posterior view. Respiratory distress and a brassy cough are the most common symptoms.

Once the diagnosis has been established, operation is mandatory. Division of the smaller arch (usually the anterior) is performed. The results are dramatic and complete relief is obtained although tracheograms indicate that considerable time is required for the cartilagenous rings to resume normal contour.

II. Right Aortic Arch with Left Ligamentum Arteriosum

A right aortic arch is not an uncommon finding and places no strain on the heart. However, the relationship of the ductus arteriosus, or if obliterated, the ligamentum arteriosum, to the aortic arch determines the presence or absence of symptoms. This structure connects the pulmonary artery with the descending aorta, originating in most cases just distal to the left subclavian artery. If it passes anterior to the trachea to connect with the arch, no compression of the trachea or esophagus will result. However, a more common course is for it to pass behind the trachea and esophagus thus forming a vascular ring which may compress the encircled structures. Again, the existence of symptoms depends on the inner circumference of this ring, and the length of the ligamentum or ductus determines this. When symptoms do exist, they are similar to those produced by a double aortic arch but are somewhat less severe. Gross[16] describes the average age of onset as about one to one and one-half years. The diagnosis is established by the same techniques as those employed in double aortic arch. Surgical therapy consists in dividing the ring in the region of the ductus, thus releasing all compression. It is important to perform this procedure as early as possible to prevent permanent tracheal deformity.

III. Anomalous Innominate Artery

This is a relatively uncommon condition which occurs when the innominate artery originates at a point further along the arch (to the patient's left) than is normal. It must wind around the anterior tracheal wall as it courses upward and to the right. If this vessel is long, the patient has no symptoms, but if it is a short structure, the tracheal compression is enough to cause symptoms. Persistent cough, wheeze, respiratory distress, and repeated respiratory infections are the resulting symptoms. Tracheograms may reveal a long anterior oblique indentation. Since one cannot divide this vital structure, relief is obtained by mobilizing and anchoring this vessel away from the trachea. It may be attached to the chest wall with several interrupted sutures placed through only the adventitia of the vessel.

IV. Anomalous Left Common Carotid Artery

The vessel is normal except that it originates from the arch more proximally (more to the patient's right) than is normal. It winds around the anterior tracheal wall as it courses upward and to the left. A respiratory stridor, recurrent pneumonia, and some respiratory distress are the resulting symptoms. Tracheogram should reveal the characteristic anterior indentation. Treatment consists of adequate mobilization and anchoring of this vessel away from the trachea rather than its division.

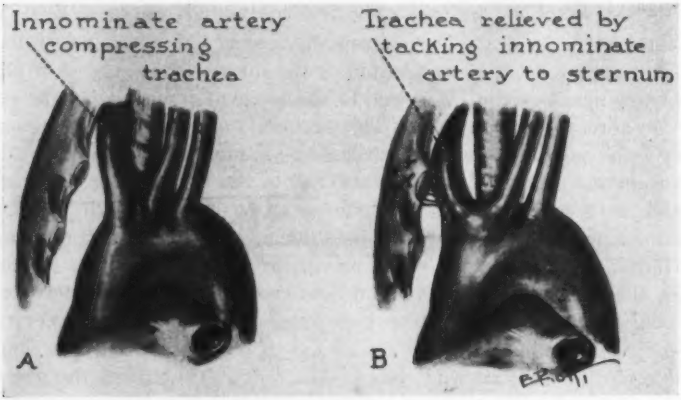

FIG. 10. Anomalous innominate artery which compresses front of trachea. A. Anatomic peculiarity of innominate artery, in which vessel has origin from aortic arch farther to patient's left than normal. As it courses upward toward right apex of chest, vessel winds around and indents front of trachea. B. Surgical correction of condition by pulling vessel forward so that it no longer touches trachea. (*From Robert E. Gross and E. D. B. Neuhausser: "Compression of the Trachea or Esophagus by Vascular Anomalies." Pediatrics, January 1951. Courtesy of Charles C Thomas, Publisher, Springfield, Illinois.*)

V. Aberrant Subclavian Artery

This is an extremely common entity in which the vessel originates independently from the left side of the aortic arch and courses upward and to the left, passing behind the esophagus. The majority of the patients are asymptomatic. The trachea is not disturbed. The diagnosis is established by a lateral x-ray view of the barium swallow of the esophagus showing the characteristic indentation of the posterior wall. Surgical therapy consists of division of this aberrant vessel.

All of these anomalous vessels except the aberrant subclavian artery cause some respiratory difficulties resulting from tracheal compression. Cough, stridor respiratory distress, and repeated pneumonia are common.

These infants obtain some relief from these difficulties by holding their heads in hyperextension. This forces the constricting vessel away from the trachea. Respiratory distress is more marked during feedings.

Tracheograms indicate that these anomalies should be corrected early to allow normal tracheal growth and to prevent permanent cartilagenous deformity.

Gross[16] emphasizes that at operation, "in all of these conditions, great care must be exercised to determine the exact anatomic relationship of the

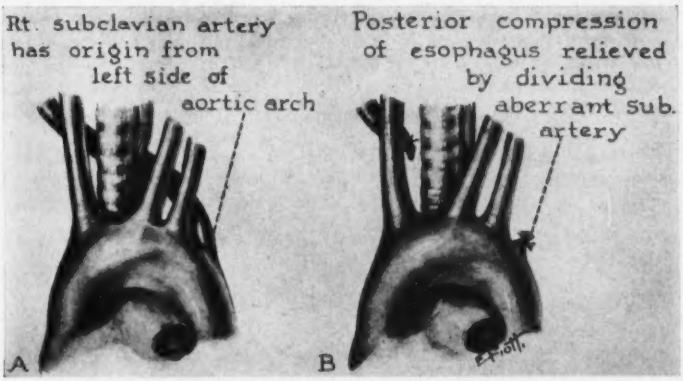

FIG. 11. Aberrant right subclavian artery. A. Anatomic arrangement. Instead of arising from innominate artery, right subclavian branches from distal part of aortic arch and runs behind esophagus, upward and toward patient's right. B. Surgical correction of condition, by double ligation and division of subclavian. (*From Robert E. Gross and E. D. B. Neuhausser: "Compression of the Trachea or Esophagus by Vascular Anomalies." Pediatrics. January 1951. Courtesy of Charles C Thomas, Publisher, Springfield, Illinois.*)

arch and all regional vessels so that exact components of an anomalous situation are known before any structure is divided." Prompt and dramatic relief follows surgical therapy.

SUMMARY

1. Some of the more commonly encountered acyanotic congenital cardiovascular anomalies are discussed.
2. Diagnostic methods are outlined.
3. Indications for surgical therapy are reviewed.

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